Slc26a2 Cas9-CKO *Strategy*

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Design Date: 2019-7-22

Project Overview

Project Name	***Slc26a2***
Project type	**Cas9-CKO**
Strain background	**C57BL/6JGpt**

Conditional Knockout strategy

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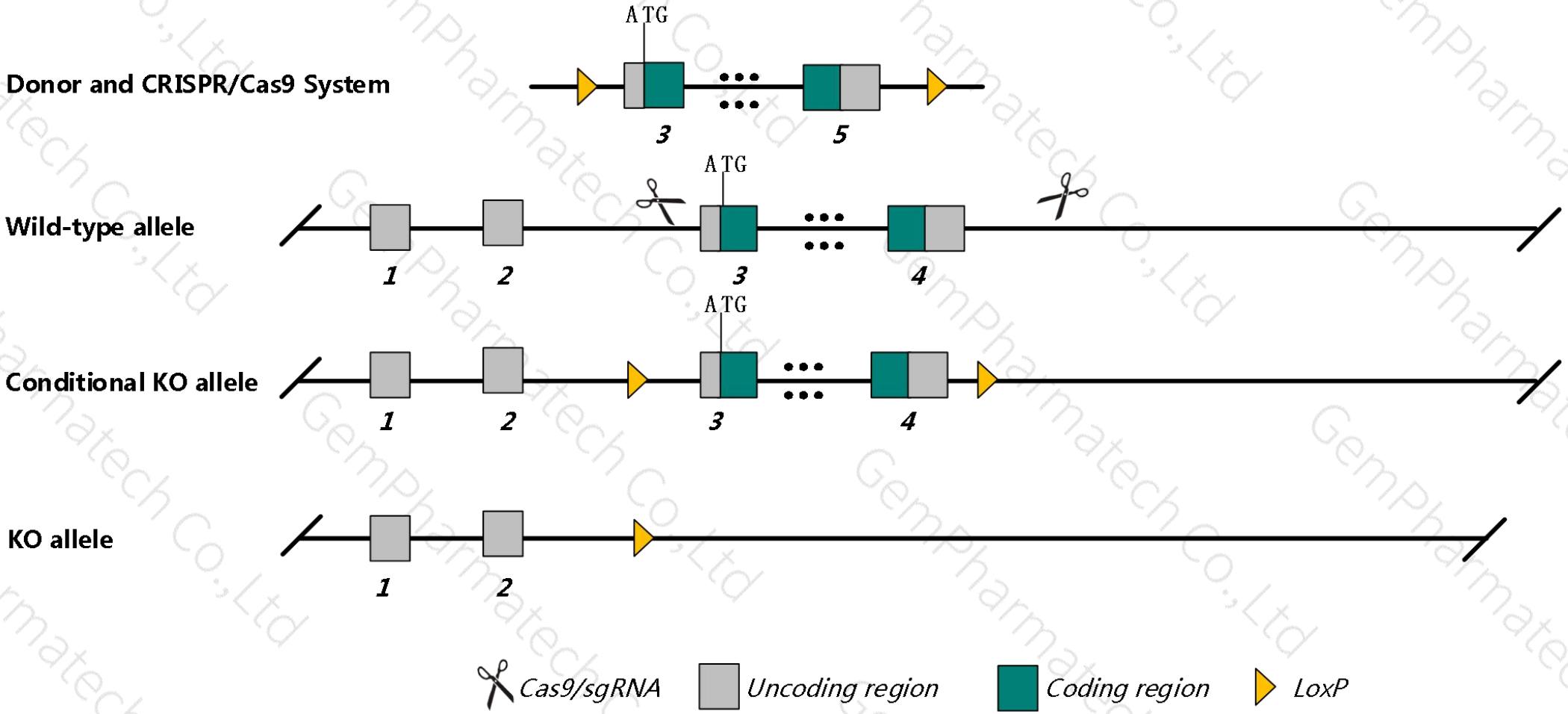
This model will use CRISPR/Cas9 technology to edit the *Slc26a2* gene. The schematic diagram is as follows:



Technical routes

- The *Slc26a2* gene has 4 transcripts.According to the structure of *Slc26a2* gene, exon3-4 of *Slc26a2*-202 (ENSMUST00000146409.7) transcript is recommended as the knockout region.The region contains all coding sequence.Knock out the region will result in disruption of protein function.

- In this project we use CRISPR/Cas9 technology to modify *Slc26a2* gene. The brief process is as follows: gRNA was transcribed in vitro, donor was constructed.Cas9, gRNA and Donor were microinjected into the fertilized eggs of C57BL/6JGpt mice.Fertilized eggs were transplanted to obtain positive F0 mice which were confirmed by PCR and sequencing. A stable F1 generation mouse model was obtained by mating positive F0 generation mice with C57BL/6JGpt mice.

- The flox mice will be knocked out after mating with mice expressing Cre recombinase, resulting in the loss of function of the target gene in specific tissues or cell types .

Notice

- According to the existing MGI data，Mice homozygous for a knock-in allele exhibit premature death, stunted growth, joint contractures, and skeletal dysplasia including kyphosis, shorter osteoporotic long bones, aberrant chondrocyte size, delayed endochondral bone ossification, and impairedchondrocyte proliferation and sulfate uptake.
- The *Slc26a2* gene is located on the Chr18. If the knockout mice are crossed with other mice strains to obtain double gene positive homozygous mouse offspring, please avoid the two genes on the same chromosome.
- This Strategy is designed based on genetic information in existing databases.Due to the complexity of biological processes, all risk of the loxp insertion on gene transcription, RNA splicing and protein translation cannot be predicted at the existing technology level.

Gene information （NCBI）

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Slc26a2 solute carrier family 26 (sulfate transporter), member 2 [*Mus musculus* (house mouse)]

Gene ID: 13521, updated on 2-Jul-2019

Summary

Official Symbol	Slc26a2 provided by MGI
Official Full Name	solute carrier family 26 (sulfate transporter), member 2 provided by MGI
Primary source	MGI:MGI:892977
See related	Ensembl:ENSMUSG00000034320
Gene type	protein coding
RefSeq status	VALIDATED
Organism	Mus musculus
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	Dtd; ST-OB
Expression	Biased expression in colon adult (RPKM 28.9), large intestine adult (RPKM 11.3) and 13 other tissues See more
Orthologs	human all

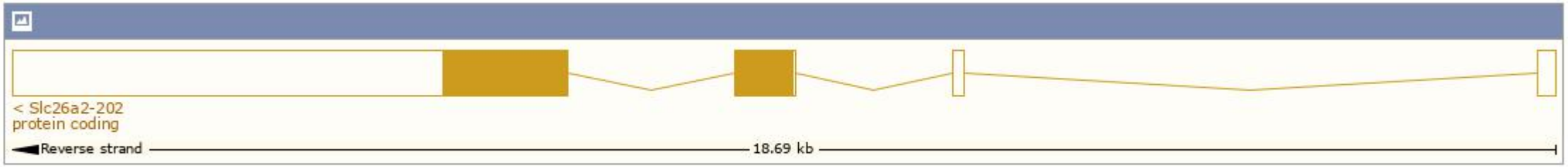
Transcript information（Ensembl）

The gene has 4 transcripts, and all transcripts are shown below:

Show/hide columns (1 hidden) Filter

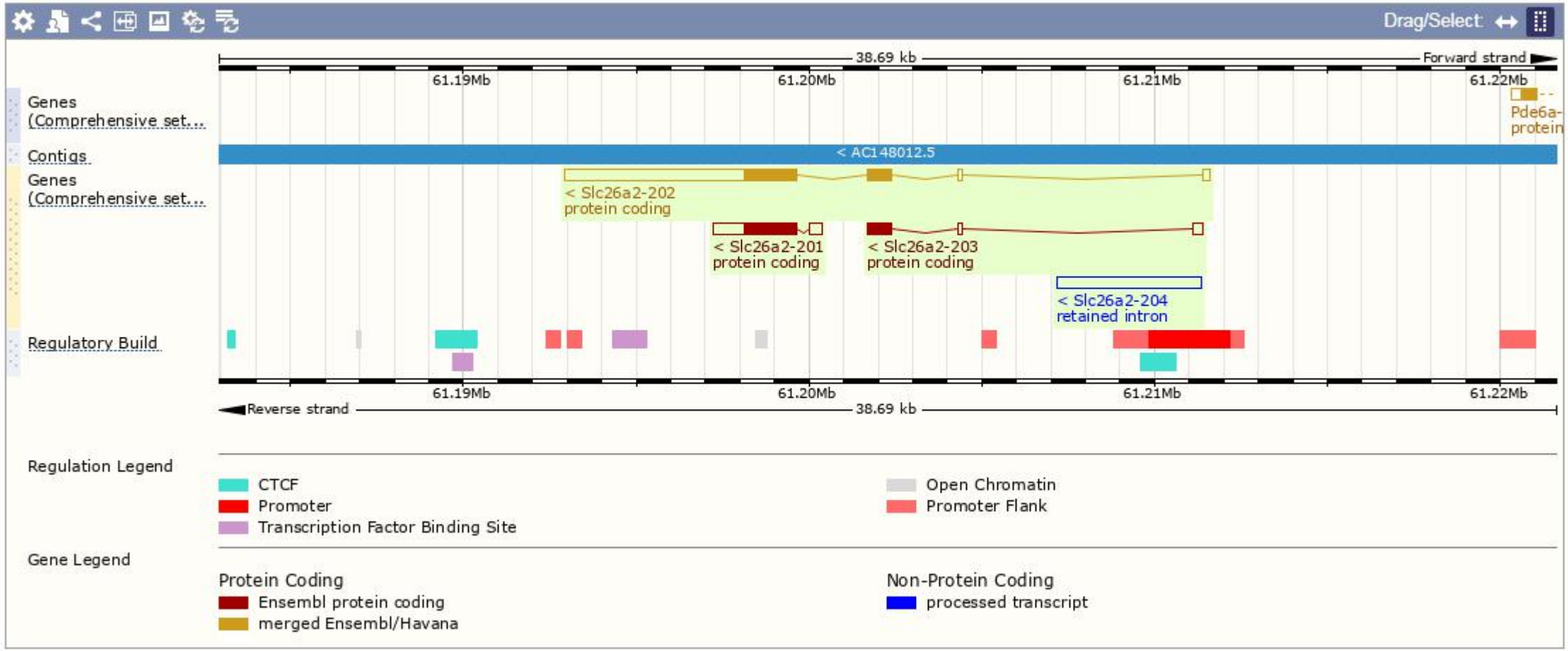
Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Slc26a2-202	ENSMUST00000146409.7	7814	739aa	Protein coding	CCDS29282	Q62273	TSL:1 GENCODE basic APPRIS P1
Slc26a2-201	ENSMUST00000037603.7	2802	504aa	Protein coding	-	F8WI08	TSL:1 GENCODE basic
Slc26a2-203	ENSMUST00000148829.1	1101	225aa	Protein coding	-	E9PZ06	CDS 3' incomplete TSL:1
Slc26a2-204	ENSMUST00000181997.1	4168	No protein	Retained intron	-	-	TSL:NA

The strategy is based on the design of *Slc26a2*-202 transcript,The transcription is shown below

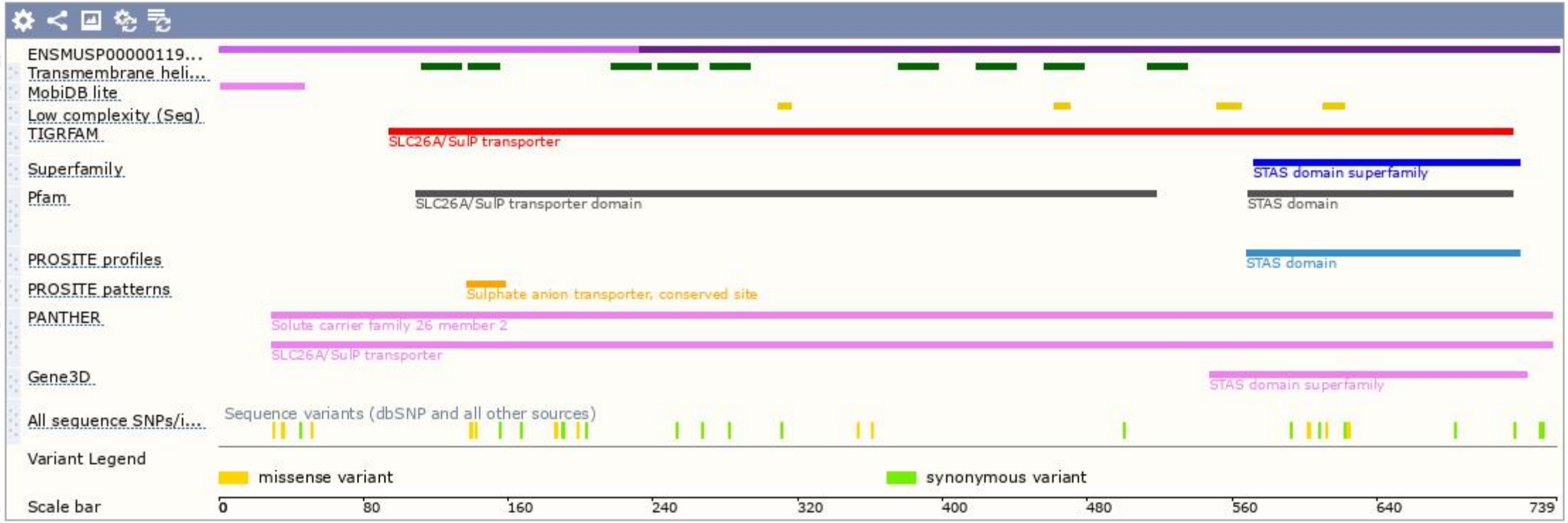


Statistics **Exons:** 4, **Coding exons:** 2, **Transcript length:** 7,814 bps, **Translation length:** 739 residues

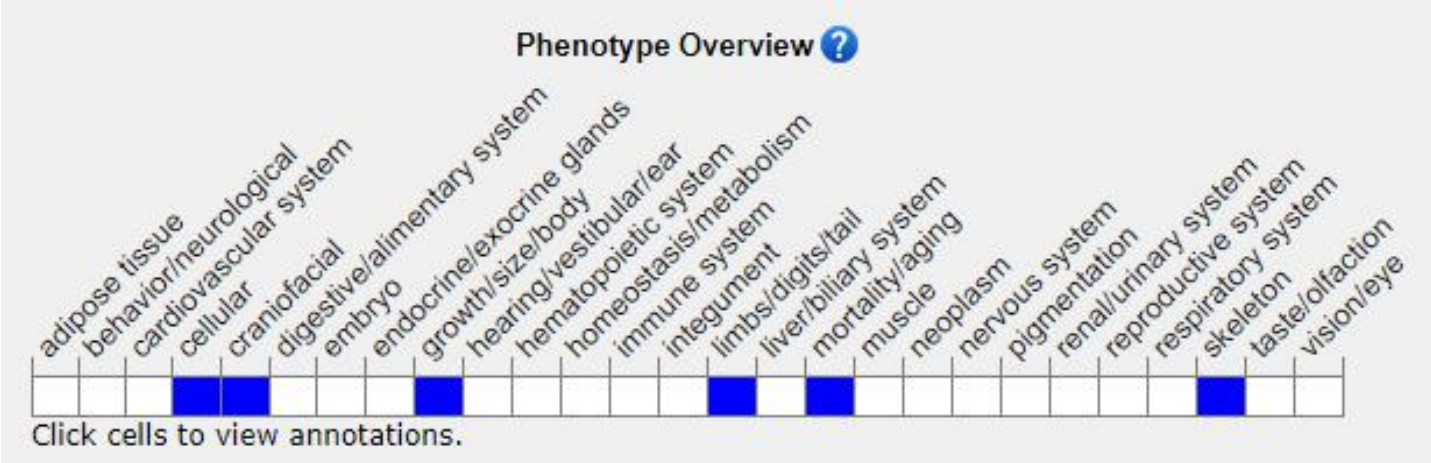
Genomic location distribution



Protein domain



Mouse phenotype description(MGI)



Phenotypes affected by the gene are marked in blue.Data quoted from MGI database(http://www.informatics.jax.org/) .

Mice homozygous for a knock-in allele exhibit premature death, stunted growth, joint contractures, and skeletal dysplasia including kyphosis, shorter osteoporotic long bones, aberrant chondrocyte size, delayed endochondral bone ossification, and impairedchondrocyte proliferation and sulfate uptake.

If you have any questions, you are welcome to inquire.
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